



New immunization law streamlines exemption process

The 2015 West Virginia Legislature enacted Senate Bill 286 relating to compulsory immunizations of school-age children. Although the bill did not weaken the State's strong immunization requirements, it did change some processes.

The new law streamlines the medical exemption review process. It moves that process from the local health departments to the West Virginia Department of Health and Human Resources (WVDHHR), where the Immunization Officer (a licensed physician) will make determinations on requests for medical exemptions. Persons who feel they are adversely affected can then appeal for review by the State Health Officer. The final determination of the State Health Officer can then be appealed through circuit court.



The bill clarifies and codifies (from interpretive rule into State Code) immunization requirements for child care centers and private schools in West Virginia in addition to public schools. The law also removes religious exemptions from child care centers' compulsory immunization requirements. Under the new law, a child entering school or a state-regulated child care center must be immunized against

(See *Immunizations*, page 11)

Statewide Disease Facts & Comparisons

A quarterly publication of the West Virginia Office of Epidemiology & Prevention Services

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Too many vaccines?

*Commentary by Dominic Gaziano, MD,
Medical Director, West Virginia Division of TB Elimination*

Opinions generated by talk show divas that immunizations lead to disease have received considerable attraction in recent years. A basic unsubstantiated view is that there are excessive numbers of immunizations with which our body is unable to contend, and that the number and variety of immunizations produce serious adverse effects and disease. The principal culprit, Dr. Andrew Wakefield, opted an opinion in the *Lancet* that fraudulently presented and although very clearly deemed false, continues to impugn the vast public health dividends that vaccines afford the American population. The scientific fact is that our immune system has infinite reserve and is called upon daily to respond to a myriad of foreign agents that approach us through our lungs and intestinal tract. This process is little understood.

A far more important question that we need to answer is: Why don't we have more vaccines? The ancient diseases of tuberculosis and malaria remain worldwide scourges. Newer diseases such as AIDS, Hepatitis C, and Ebola are now members of the disease apocalypse. A question then arises: Why are these diseases failing to be controlled by public health immunizations?

For the past one hundred years, attempts to control tuberculosis through immunological processes have been widely a failure. Dr. Robert Koch, soon after his discovery of the tubercle bacilli, unsuccessfully offered tuberculin as a vaccine. In 1921, the BCG was developed based upon the success with the smallpox vaccination that attenuated mycobacteria would have been protective. This hope for control failed to produce significant results except to diminish infant central nervous system tuberculosis. Multiple attempts to vaccinate against this worldwide scourge as recent as February 2013 failed to provide protection. These failures are not wholly unanticipated. The TB germ, despite the best of our innate immunity, can live unmolested for decades in the body. Even a case of

successfully treated tuberculosis cannot fend off a new infection.

Malaria, a disease eliminated in temperate climates with mosquito eradication programs, remains widespread in tropical and semi-tropical climates. It seems that a net is the best we can do for malaria. A malaria vaccine appears to be a complex process since its intended target is a more complicated protozoan. This factor, with complicated life cycles, represents a problem for vaccine development.

Hepatitis C seems to be a special scourge in West Virginia. It is transmitted largely as a result of a burgeoning drug culture. Amazingly, a shared needle or a nasal aspiration straw is all that is required for this disease to be transmitted. Despite the conquest of Hepatitis A and B, Hepatitis C remains a large and increasing problem in our State. It appears that the Hepatitis C virus is highly variable and has strains that rapidly mutate. These facts make the development of an effective vaccine very difficult. The recent development of an effective treatment - at nearly \$80,000 per patient - can only represent a partial answer to this condition.

It appears that sex is a very effective method of transmitting some diseases. Interestingly, syphilis brought to Europe by Christopher Columbus and AIDS with its limited entry into the population, were thoroughly transmitted within 30 years. Human

immunodeficiency virus (HIV) - the leading cause of an infectious disease death - procures immunization for this pandemic to be halted. Natural immunity does not seem to occur since there is almost no recovery of AIDS patients. If cured, "HIV does not retain antigenicity which presents a problem for vaccine development."

The recent terror from Ebola, which is widespread in Western Africa, cries for a vaccine. Currently, the medication given early in Ebola seems to have a high cure rate of approximately 85%. There is also an expectation in a phase one clinical trial of an Ebola vaccine that may be the answer to this pandemic. The vaccine seems to be well-tolerated and produces a healthy immune response.

Yes: These aforementioned diseases, not the vaccines, have collectively resulted in billions of deaths and affliction worldwide. Yes, TV talk shows: We need more vaccines to control the old and new diseases that are continuing to exact their terrible tolls on our society. ❖



La Crosse virus detected and isolated from invasive mosquito species

By Eric Dotseth and Miguella Mark-Carew,
Division of Infectious Disease Epidemiology

La Crosse virus (LACV), a member of the genus *Orthobunyavirus* family Bunyaviridae, has become one of the most common causes of pediatric arboviral encephalitis in the United States. La Crosse encephalitis has traditionally been associated with forested ecosystems in the upper mid-western United States but more recently has emerged in states in the Appalachian region, such as West Virginia, Tennessee, North Carolina, and Virginia. LACV is maintained in nature through horizontal transmission to sciurid rodents, particularly chipmunks and squirrels, and vertical transmission, from female mosquitoes to their offspring.

The primary mosquito vector of LACV, the eastern tree-hole mosquito (*Aedes triseriatus*) is found in the eastern United States; however, two invasive species, the Asian tiger mosquito (*Aedes albopictus*), and the Asian bush mosquito (*Aedes japonicus*), have recently emerged in the Appalachian region. Both invasive species have been shown experimentally to be competent LACV vectors (Grimstad et al. 1989; Sardelis, Turell & Andre 2002). Viable LACV capable of causing human infection has also been isolated from *Aedes albopictus* collected in the field (Gerhardt et al. 2001). Until recently, LACV RNA has only been detected in field-collected *Aedes japonicus* (Westby et al. 2011).

In a recent study published in *Emerging Infectious Diseases*, scientists from the Virginia Polytechnic Institute and State University and the WVDHHR detected and isolated LACV from *Aedes japonicus* collected from southern West Virginia and southwestern Virginia. In this study, LACV RNA was detected in a sample of *Aedes japonicus* mosquitoes reared from eggs collected in Wise County, Virginia in August 2005 and one adult *Aedes japonicus* sample collected from Montgomery County,

Virginia in July 2008. In 2013, LACV RNA was detected in five samples of adult *Aedes japonicus* collected from three West Virginia counties (Fayette, Kanawha, Cabell) (Harris et al. 2015). In 2009, adult *Aedes japonicus* were tested for infectious LACV by inoculating Vero cells with mosquito homogenate. Active LACV was isolated from two mosquito samples; one collected in Montgomery County, Virginia and the other captured from neighboring Craig County, Virginia (Harris et al. 2015).

The Asian bush mosquito, *Aedes japonicus*, could increase LACV human burden in the future. The distribution of *Aedes japonicus* is expanding rapidly across the United States (Kampen & Werner 2014). This rapid spread of *Aedes japonicus* is likely related to its cold hardiness (extending its activity into early spring and late fall) and ability to develop in different larval habitats (Kaufman & Fonseca



Asian bush mosquito (*Aedes japonicus*)
Photo by Ary Faraji

2014). Unlike the primary LACV mosquito vector *Aedes triseriatus*, *Aedes japonicus* will develop in sunlit environments near human habitats (Joy & Sullivan 2005). Also, *Aedes japonicus* has a feeding preference for mammals (Molaei et al. 2008), including human blood-feeding tendencies (Molaei et al. 2008; Molaei et al. 2009).

La Crosse encephalitis can be managed through source reduction and personal protection.

Like other LACV mosquito vectors, *Aedes japonicus* multiply in containers that hold water. *Aedes japonicus* larvae develop in tree holes, tires, drain pipes, catch basins in storm drain systems, and containers made of concrete, stone, plastic or metal. Although *Aedes japonicus* develops in tree holes, tires are a more important breeding habitat for this mosquito species (Bartlett-Healy et al. 2012). Mosquito repellents, proper clothing (long sleeve shirts, pants and socks) and secure, intact screens for doors and windows will reduce contact between humans and infectious mosquitoes.

References

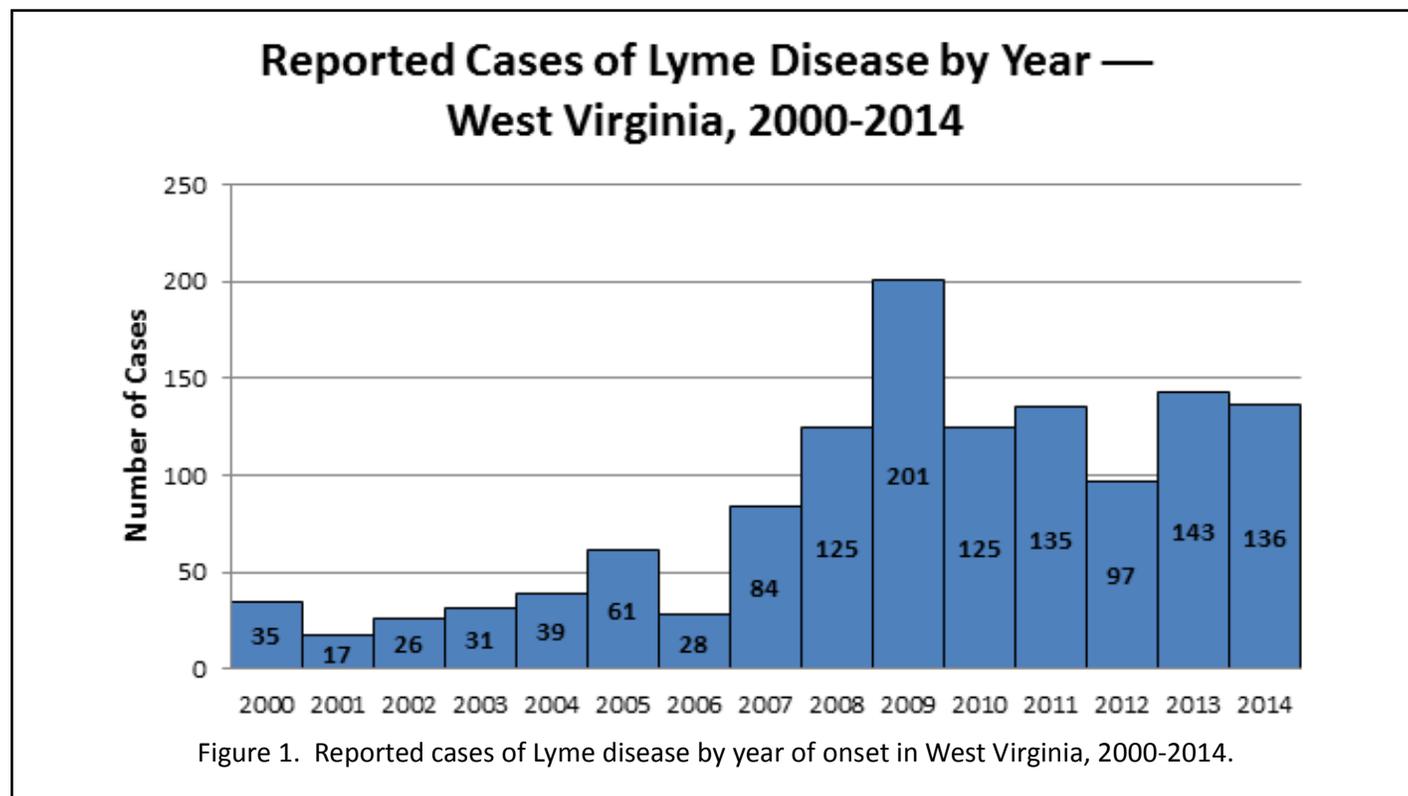
Bartlett-Healy, K., I. Unlu, P. Obenauer, T. Hughes, S. Healy, T. Crepeau, A. Farajollah, B. Kesavaraju, D. Fonseca, G. Schoeler, R. Gaugler & D. Strickman. 2012. Larval mosquito

(See **LaCrosse**, page 11)

Analyzing the emergence of Lyme disease and its vector, the blacklegged tick, in West Virginia

By Miguella Mark-Carew and Eric Dotseth, West Virginia Division of Infectious Disease Epidemiology

Lyme disease, named for a town in Connecticut, has become an important tickborne disease across the United States. In West Virginia, it is the most common vectorborne disease reported. The incidence of Lyme disease in the State has been steadily increasing over the past fifteen years (Figure 1, below) and likely reflects an expansion of *Ixodes scapularis*, the tick vector associated with transmitting *Borrelia burgdorferi*, the bacteria that causes Lyme disease.

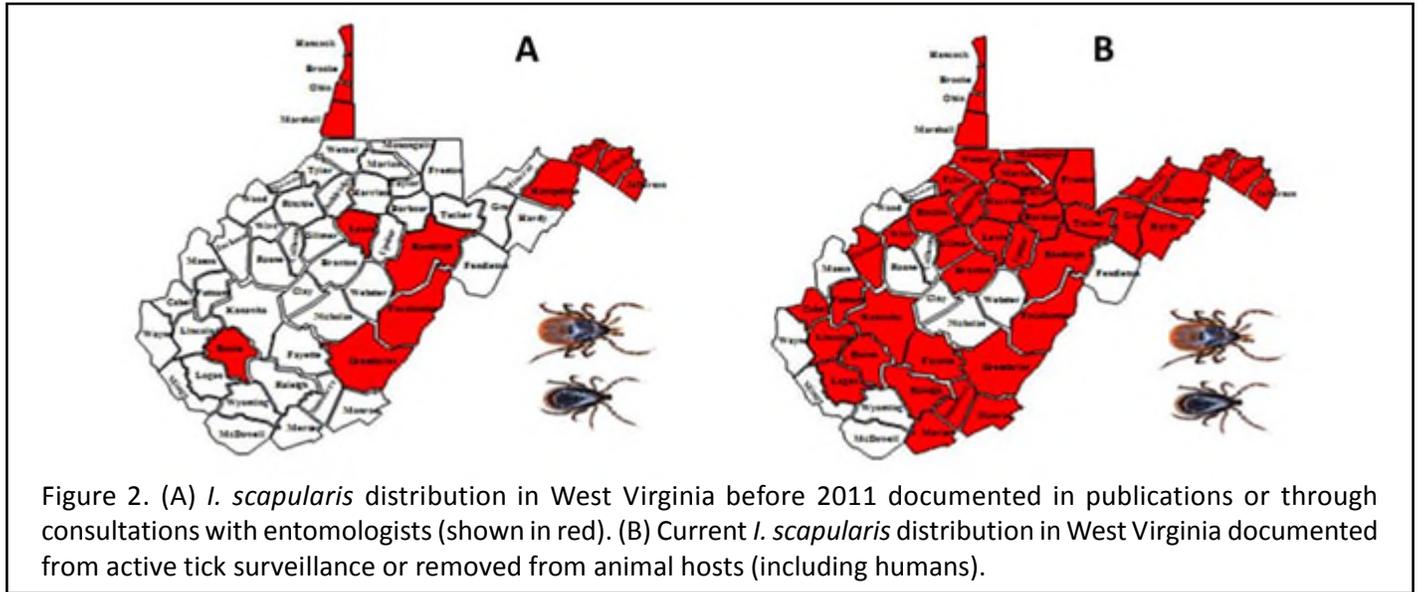


The known distribution of *I. scapularis*, also known as the “blacklegged tick” or “deer tick,” was limited to West Virginia’s eastern panhandle (Hall et al. 1991; Dennis et al. 1998; Culik et al. 1999; Diuk-Wasser et al. 2010; Amrine, pers. comm.) with occasional occurrences in other eastern counties and West Virginia’s northern panhandle (Amrine, pers. comm., Crutchfield, pers. comm.) (Figure 2A, page 5). Most counties in the western part of the State were not considered suitable habitat for blacklegged ticks (Brownstein, Holford & Fish 2003; Brownstein, Holford & Fish 2005; Diuk-Wasser et al. 2010; Diuk-Wasser et al. 2012), and the risk for Lyme disease or human anaplasmosis was predicted to be low in most of the State (CDC 1999; Wimberly, Baer & Yabsley 2008).

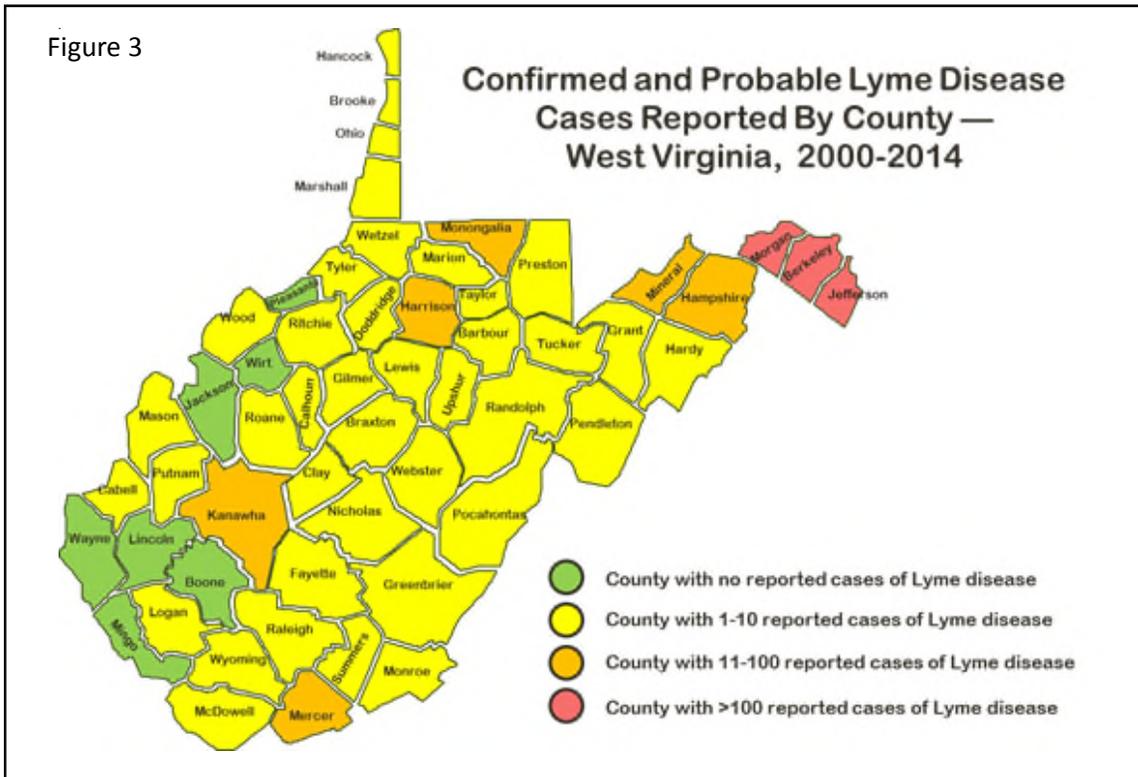
WVDHHR began actively searching for blacklegged tick populations in the State in 2011. In cooperation with the West Virginia Division of Natural Resources and local veterinarians, blacklegged ticks have now been identified in most counties across the State (Figure 2B, page 5). The emergence of blacklegged ticks and Lyme disease across West Virginia during the past few years has also been documented in western Pennsylvania (Pennsylvania Dept. of Health, 2015) and eastern Ohio (McKinnon 2012; Wang et al. 2014).

(See *Lyme Disease*, page 5)

(Lyme Disease, continued from page 4)



Lyme disease incidence in humans is following the westward expansion of the blacklegged ticks. Most human Lyme disease cases have occurred in West Virginia’s eastern panhandle; Berkeley, Jefferson and Morgan counties account for almost 75% of Lyme disease cases reported from 2000-2014 (Figure 3, below). Fewer human cases have been reported from counties in central West Virginia, and some counties in the western part of the State have not reported human Lyme disease cases.



(See *Lyme Disease*, page 10)

West Virginia Infectious Disease Outbreak Report, January-March 2015

Introduction:

In West Virginia, outbreaks are immediately reportable to local health departments (LHDs), regardless of setting, as per Reportable Disease Rule 64CSR7. LHDs, in collaboration with the West Virginia Bureau for Public Health, Division of Infectious Disease Epidemiology (DIDE) investigate all reported outbreaks. DIDE provides outbreak surveillance reports on a monthly and annual basis, and upon request. This report provides a brief description of confirmed outbreaks during the first quarter of 2015. All data provided are provisional, since several investigations are ongoing.

Methods:

Data on outbreaks are routinely compiled in Microsoft Excel 2010. Data analyzed for the purpose of this report include information on outbreak type and setting, reporting region, time of reporting to LHDs and DIDE by region, clinical diagnosis, and laboratory information.

Results:

During the months of January, February, and March 2015, there were 105 outbreaks reported in West Virginia. Of the 105 reported outbreaks, 101 (96%) were confirmed as outbreaks or clusters of disease. Four (4) were investigated and determined not to be outbreaks or clusters. Eighty-one (81) were reported from healthcare facilities, 12 from schools, 3 from daycares, 2 from hotels, 1 from a residential center, 1 from a social club, and 1 from a correctional facility.

Among the 81 healthcare-associated outbreaks reported, 74 were from long-term care facilities (LTCFs), 5 from assisted living facilities, 1 from a hospital, and 1 from a rehabilitation center.

Respiratory Illness Outbreaks from January-March 2015 (n=65)

| Type of Outbreak or Cluster | Number of Outbreaks | Outbreak Setting | Laboratory Testing |
|-----------------------------------|---------------------|---------------------------|--|
| Influenza | 58 | 46 LTCFs | 10 PCR* Confirmed 36 RIDT** Confirmed |
| | | 5 Schools | RIDT** Confirmed |
| | | 3 Assisted Living | 1 PCR* Confirmed 2 RIDT** Confirmed |
| | | 2 Daycares | RIDT** Confirmed |
| | | 1 Residential Facility | RIDT** Confirmed |
| | | 1 Rehabilitation Facility | RIDT** Confirmed |
| Acute Respiratory Illness | 2 | 2 LTCFs | Negative or Non-contributory |
| Influenza-Like Illness | 2 | 1 Correction Facility | Not Done |
| | | 1 School | Not Done |
| Pertussis | 2 | Schools | PCR* Confirmed |
| Respiratory Syncytial Virus (RSV) | 1 | Daycare | PCR* Confirmed |

* PCR: Polymerase Chain Reaction ** RIDT: Rapid Influenza Diagnostic Test

(See **Outbreaks**, page 7)

(Outbreaks, continued from page 6)

Enteric Disease Outbreaks from January-March 2015 (n=30)

| Type of Outbreak or Cluster | Number of Outbreaks | Outbreak Setting | Laboratory Testing |
|-----------------------------|---------------------|-------------------|--------------------|
| Acute Gastroenteritis | 19 | 14 LTCFs | Not Done |
| | | 3 Schools | Not Done |
| | | 1 Social Club | Not Done |
| | | 1 Assisted Living | Not Done |
| Norovirus Gastroenteritis | 8 | 8 LTCFs | PCR* Confirmed |
| Rotavirus Gastroenteritis | 2 | 1 LTCF | PCR* Confirmed |
| | | 1 Assisted Living | PCR* Confirmed |
| Salmonellosis | 1 | Hospital | PCR* Confirmed |

* PCR: Polymerase Chain Reaction

Rash Outbreaks from January-March 2015 (n=5)

| Type of Outbreak or Cluster | Number of Outbreaks | Outbreak Setting | Laboratory Testing |
|-----------------------------|---------------------|------------------|--------------------|
| Undetermined Rash Illness | 2 | Hotel Pool | Not Done |
| Scabies | 2 | LTCFs | Not Done |
| Varicella | 1 | School | Not Done |

Multidrug-Resistant Organism (MDRO) Outbreaks from January-March 2015 (n=1)

| Type of Outbreak or Cluster | Number of Outbreaks | Outbreak Setting | Laboratory Testing |
|------------------------------|---------------------|------------------|--------------------|
| <i>Clostridium difficile</i> | 1 | LTCF | Lab Confirmed |

(See **Outbreaks**, page 8)

(*Outbreaks*, continued from page 7)

All surveillance regions reported outbreaks during this period (Figure 1). Respiratory outbreaks were the most common reported during this period, accounting for 65 (64%) of all confirmed outbreaks (Figure 2). The peak incidence of respiratory outbreaks occurred in January (Figure 3, page 9). The majority of respiratory outbreaks 58 (89%) were caused by influenza. Enteric outbreaks represented the second most common outbreaks 30 (30%). Of the 30 enteric disease outbreaks, only 11 (36%) had laboratory confirmation.

Figure 1. Confirmed Outbreaks by Region, West Virginia, January-March 2015 (n=101)

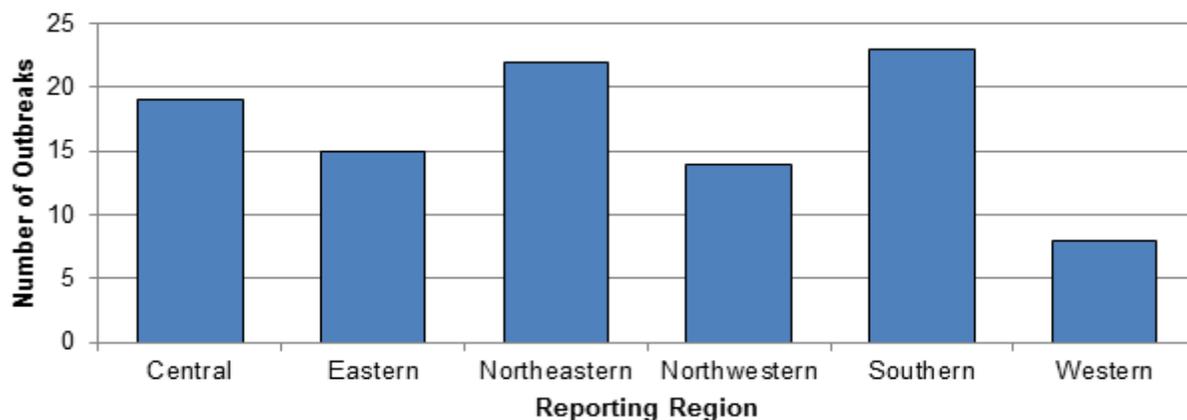
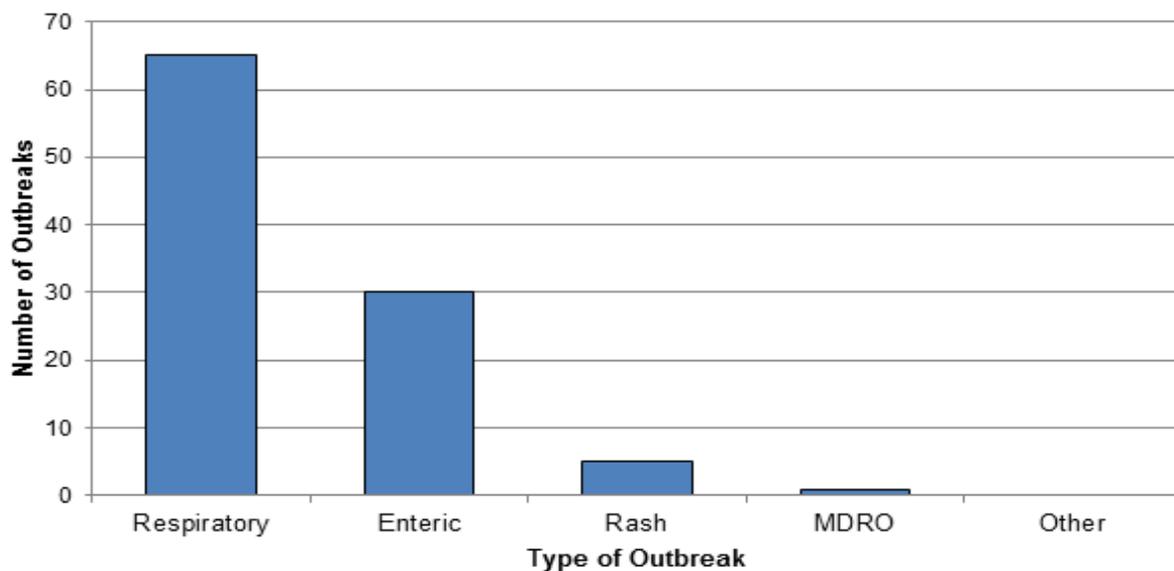
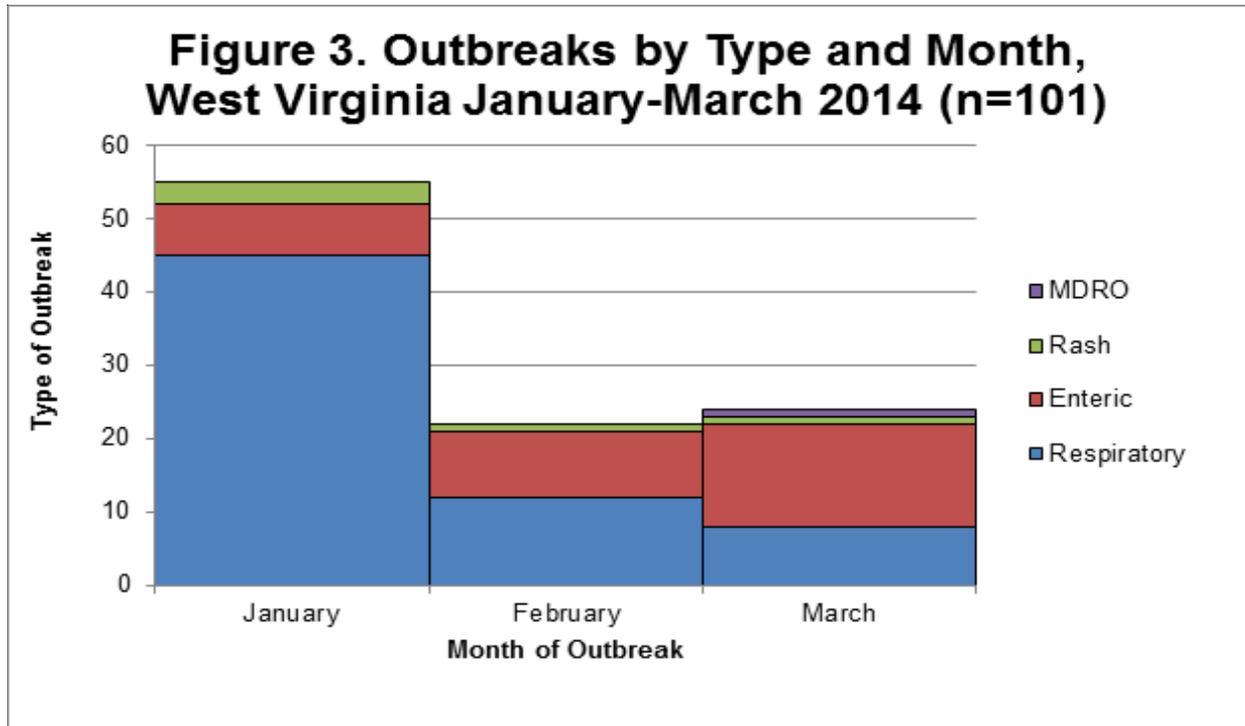


Figure 2. Confirmed Outbreaks by Type in West Virginia, January-March 2015 (n=101)



(See *Outbreaks*, page 9)

(Outbreaks, continued from page 3)



Conclusions:

There was marked increase in influenza outbreaks during this period which suggests a moderately severe influenza season. Influenza surveillance data indicate that flu season peaked in WV at the beginning of January. The predominant strain of influenza circulating in WV was influenza A H3N2. Nationally, CDC reports that more hospitalizations and deaths are typical of H3N2 seasons, which tend to disproportionately impact young children and older people. In addition, nearly 80% of the H3N2 viruses circulating this season were different from the vaccine virus. The predominance of drifted H3N2 viruses is probably responsible for the reduced protection offered by this season's vaccine and may also have contributed to the severity of the season. The reduced protection offered by the flu vaccine this season makes the appropriate use of antiviral medications especially important among high-risk populations.

The majority of influenza outbreaks were reported from healthcare facilities, specifically LTCFs. This underscores the importance of preseason preparations in LTCFs, such as influenza vaccination of residents and staff, standing orders for chemoprophylaxis during outbreak situations, and training and education on hand hygiene and respiratory etiquette.

DIDE encourages LHDs to provide testing options to facilities during enteric outbreaks. The West Virginia Office of Laboratory Services provides free testing for enteric pathogens during outbreaks.

For information on outbreak guidelines or any disease or condition, please visit the Division of Infectious Diseases Epidemiology's website at www.dide.wv.gov or call (304) 558-5358, or toll-free in West Virginia (800) 423-1271. ☒

**National
HIV TESTING Day
JUNE 27**

**Free, confidential HIV testing is available near you. To find out more, call the West Virginia HIV/AIDS Hotline:
1-800-642-8244**

(Lyme Disease, continued from page 5)

Blacklegged ticks can be active year-round but are more likely to bite and transmit Lyme disease during warmer months (April through September). Lyme disease prevention primarily focuses on avoiding tick bites. Wooded or bushy areas with high grass or extensive leaf litter are conducive to ticks carrying Lyme disease. Conducting a full-body tick check and bathing after returning from tick-infested areas will reduce the potential of Lyme disease transmission; an infected tick has to be attached for a long period of time (upwards of 24 hours) to transmit Lyme disease. Tick prevention measures should also be applied to pets which can serve as unwilling sources of blacklegged ticks.



A blacklegged tick having lunch.

References

- Amrine, J. 2015. Professor Emeritus, Division of Plant & Soil Sciences, West Virginia University, Morgantown, WV.
- Brownstein, J. S., T. R. Holford & D. Fish. 2003. A climate-based model predicts the spatial distribution of the Lyme disease vector *Ixodes scapularis* in the United States. *Environmental Health Perspectives* 111 (9): 1152-1157.
- Brownstein, J. S., T. R. Holford & D. Fish. 2005. Effect of climate change on Lyme disease risk in North America. *Ecohealth* 2 (1): 38-46.
- CDC. 1999. Appendix methods used for creating a national Lyme disease risk map. *Morbidity & Mortality Weekly Report* 48: 21-24.
- Crutchfield, B. 2015. Plant/Pest Biologist. Pest Identification Laboratory, Agricultural Pest Survey Programs Unit, Plant Industries Division, West Virginia Department of Agriculture, Charleston, WV.
- Culik, M. P. 1999. West Virginia Tick Information. <http://www.wvu.edu/~agexten//ipm/insects/2tick.htm>. Accessed Mar. 20, 2015.
- Dennis, D. T., T. S. Nekomoto, J. C. Victor, W. S. Paul & J. Piesman. 1998. Reported distribution of *Ixodes scapularis* and *Ixodes pacificus* (Acari: Ixodidae) in the United States. *Journal of Medical Entomology* 35 (5): 629-638.
- Diuk-Wasser, M. A., A. Gatewood Hoen, P. Cislo, R. Brinkerhoff, S. A. Hamer, M. Rowland, R. Cortinas, G. Vourc'h, F. Melton, G. J. Hickling, J. I. Tsao, J. Bunikis, A. G. Barbour, U. Kitron, J. Piesman & D. Fish. 2012. Human risk of infection with *Borrelia burgdorferi*, the Lyme disease agent, in eastern United States. *American Journal of Tropical Medicine & Hygiene* 86 (2): 320-327.
- Diuk-Wasser, M. A., G. Vourc'h, P. Cislo, A. Gatewood Hoen, F. Melton, S. A. Hamer, M. Rowland, R. Cortinas, G. J. Hickling, J. I. Tsao, A. G. Barbour, U. Kitron, J. Piesman & D. Fish. 2010. Field and climate-based model for predicting the density of host-seeking nymphal *Ixodes scapularis*, an important vector of tick-borne disease agents in the eastern United States. *Global Ecology & Biogeography* 19: 504-514.
- Hall, J. E., J. W. Amrine, R. D. Gais, V. P. Kolanko, B. E. Hagenbuch, V. F. Gerencser & S. M. Clark. 1991. Parasitization of humans in West Virginia by *Ixodes cookei* (Acari: Ixodidae), a potential vector of lyme borreliosis. *Journal of Medical Entomology* 28 (1): 186-189.
- McKinnon, J. M. 2012. Number of deer ticks surging across Ohio: Insect is only carrier of Lyme disease. Toledo Blade. January 24, 2012.
- Pennsylvania Department of Health. 2015. Lyme Disease Cases and Incidence Reported in PA by County. <http://www.portal.state.pa.us/portal/server.pt?open=18&objID=1405493&mode=2>. Accessed March 20, 2015.
- Wang, P., M. N. Glowacki, A. E. Hoet, G. R. Needham, K. A. Smith, R. E. Gary & X. Li. 2014. Emergence of *Ixodes scapularis* and *Borrelia burgdorferi*, the Lyme disease vector and agent, in Ohio. *Frontiers in Cellular & Infection Microbiology* 4: 1-9.
- Wimberly, M. C., A. D. Baer & M. J. Yabsley. 2008. Enhanced spatial models for predicting the geographic distributions of tick-borne pathogens. *International Journal of Health Geographics* 7: 15. ☒

(LaCrosse, continued from page 3)

habitat utilization and community dynamics of *Aedes albopictus* and *Aedes japonicus*. *Journal of Medical Entomology* **49** (4): 813-824.

Gerhardt, R. R., K. L. Gottfried, C. S. Apperson, B. S. Davis, P. C. Erwin, A. B. Smith, N. A. Panella, E. E. Powell & R. S. Nasci. 2001. First isolation of La Crosse virus from naturally infected *Aedes albopictus*. *Emerging Infectious Diseases* **7** (5): 807-811.

Grimstad, P. R., J. F. Kobayashi, M. Zhang & G. B. Craig. 1989. Recently introduced *Aedes albopictus* in the United States – potential vector of La Crosse virus (Bunyaviridae, California serogroup). *Journal of the American Mosquito Control Association* **5**: 422-427.

Harris, M. C., E. J. Dotseth, B. T. Jackson, S. D. Zink, P. E. Marek, L. D. Kramer, S. L. Paulson & D. M. Hawley. 2015. La Crosse virus in *Aedes japonicus japonicus* mosquitoes in the Appalachian region, United States. *Emerging Infectious Diseases* **21** (4): 646-649.

Joy, J. E. & S. N. Sullivan. 2005. Occurrence of tire inhabiting mosquito larvae in different geographic regions of West Virginia. *Journal of the American Mosquito Control Association* **21** (4): 380-386.

Kampen, H. & D. Werner. 2014. Out of the bush: The Asian bush mosquito *Aedes japonicus japonicus* (Theobald, 1901)

(Diptera, Culicidae) becomes invasive. *Parasites & Vectors* **7**: 59.

Kaufman, M. G. & D. M. Fonseca. 2014. Invasion biology of *Aedes japonicus japonicus* (Diptera: Culicidae). *Annual Review of Entomology* **59**: 1-49.

Molaei, G., T. G. Andreadis, P. M. Armstrong & M. Diuk-Wasser. 2008. Host-feeding patterns of potential mosquito vectors in Connecticut, USA: Molecular analysis of bloodmeals from 23 species of *Aedes*, *Anopheles*, *Culex*, *Coquillettidia*, *Psorophora* & *Uranotaenia*. *Journal of Medical Entomology* **45** (6): 1143-1151.

Molaei, G., A. Farajollah, J. J. Scott, R. Gaugler & T. G. Andreadis. 2009. Human bloodfeeding by the recently introduced mosquito, *Aedes japonicus japonicus*, and public health implications. *Journal of the American Mosquito Control Association* **25** (2): 210-214.

Sardelis, M. R., M. J. Turell & A. R. G. Andre. 2002. Laboratory transmission of La Crosse virus by *Ochlerotatus j. japonicus* (Diptera: Culicidae). *Journal of Medical Entomology* **39** (4): 635-639.

Westby, K., C. Fritzen, J. Huang, E. Jaske, D. Paulsen, C. Jones & A. C. Moncayo. 2011. La Crosse encephalitis in eastern Tennessee: Evidence of invasive mosquito (*Aedes albopictus* and *Ochlerotatus japonicus*) involvement in the transmission of an indigenous disease. *American Journal of Tropical Medicine & Hygiene* **85** (supplement): 374. ☒

(Immunizations, continued from page 1)

chickenpox, hepatitis B, measles, meningitis, mumps, diphtheria, polio, rubella, tetanus and whooping cough or obtain a medical exemption from the Bureau for Public Health. Additionally, state-regulated child care centers will require three types of vaccination, if age appropriate, that are not required for school entry: Pneumococcal conjugate vaccine, haemophilus influenza type B (Hib), and hepatitis A vaccine.

This new law codifies all of the vaccines required for school. Currently, Tdap, hepatitis B, chickenpox, and meningitis vaccines are included in students' required shot schedules. These vaccines will now be in State Code.

The bill allows DHHR to issue future procedural rules to govern the administration of immunization requirements.

The bill changes the Childhood Immunization Advisory Committee to the Immunization Advisory Committee and adds pharmacists and three consumer members to the Committee.

The bill removes language which made it a misdemeanor for any parent to not vaccinate their children against diseases listed in State Code.

The revised immunization law goes into effect June 16, 2015.

Other specific administrative processes are expected to be set forth by the State Health Officer, Dr. Rahul Gupta, in the coming weeks through a procedural rule. ☒

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